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Body Hair Scores and Total Hair Diameters in Healthy Women in the Kırıkkale Region, of Turkey

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It was aimed to determine the normal hair scores of women of Kırıkkale region according to the Ferriman- Gallwey scale and to investigate the relationship between the hair shaft diameter and hair scores.

Hair scores were calculated in 204 healthy women, and hair shaft diameters were measured from the hair samples collected from 60 patients. Body mass index, waist to hip ratio, insulin resistance and blood androgen levels were determined.

Neutral, hormonal and total hair scores were 2.1 ± 1.4, 3.1 ± 2.7 and 5.2 ± 3.6, respectively. The average total hair diameter and hormonal hair diameter were 191.93 ± 90.49 μm and 121.8 ± 75.9 μm respectively. The correlation between total hair diameter and total hair score was statistically significant (r=0.704 $p<0.001$). Also, the correlation between hormonal hair diameter and hormonal hair score was statistically significant (r=0.724 $p<0.001$). While hair scores and diameters show meaningful positive correlation with androgen levels, they show negative correlation with age.

In our population, 95% value of total hair score was 11, and for the hormonal score, it was 9. Hair diameters increase with hair score, regardless of total or hormonal of hair scores. Hair scores and hair diameters may be affected by blood androgens in healthy women.

Key Words: Hirsutism, hair scores, hair diameters, androgen

INTRODUCTION

Social and clinical reactions to hirsutism may vary significantly. Androgen-dependent hair (excluding the pubic and axillary hair) occurs in only 5% of premenopausal Caucasian women and is considered abnormal in Caucasian women of North America. However, the presence of terminal hair in androgen-dependent hair, excluding the pubic and axillary hair, is viewed as normal and is socially acceptable in some ethnic groups, such as Eskimos and the people of the Mediterranean origin.[1]

Clinicians must view hirsutism as both an endocrine problem and a cosmetic problem. To the affected women, hair growth on the face or on another area is disturbing on several levels. It may be caused by a certain disease. Sexuality may change. Social acceptance may alter. Fertility may be impaired.[2] Hirsutism is more than a cosmetic problem and can psychologically handicap women.[3]

Clinicians must distinguish normal biological variations from cases of abnormally hirsute women. In 20% of all hirsute women, androgen excess is only found locally at the level of the hair follicle; that is hirsutism is idiopathic. Important causes of excessive androgen, such as an ovarian tumor, need to be excluded.[4,5] Clinical problems related to hirsutisms in women have usually been approached as if there was a clear line dividing hirsutism and the normal state. However, there is no such as distinction causing an additional clinical problem.

Different regions are affected in a different manner. For example, in one patient, the face and breasts might be affected and in another the lower abdomen and thighs might be affected. Five gradings based on densities and areas involved,

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were determined for each of 11 sites by Ferriman and Gallwey.[6] Hair growth was scored according to the sum of the grading obtained. They obtained a semi-quantitative method for clinical evaluation of body hair.[6]

In this prospective study, the determination of body hair scores and total hair diameters of healthy women of Kırıkkale region was targeted. Parameters affecting hair scores and diameters were investigated.

MATERIALS AND METHODS

We studied 204 healthy women, attending Kırıkkale University Obstetric and Gynecology clinic as outpatients for check-up. Patients taking drugs, that have adverse effects on hair growth, were excluded from the study. None of our patients were experiencing menopause. Informed consent was obtained from all the patients. Two observers scored the hair scores independently. The patients' ages ranged from 20 to 54. According to the Ferriman-Gallwey scale, the body hair score was measured in 11 different areas (lip, chin, chest, upper back, sacro- iliac region, upper and lower abdomen, arm and back of forearm, thigh and leg); only terminal hair growth was considered and graded from zero to four. The sum of the grading on the forearms and legs has been termed the "indifferent e.g. neutral" score and the sum of the grading from all other sites as the "hormonal" score.[6] Eight was supported as the threshold between hirsutism and normal hormonal score.[6] In 60 cases that accepted hair measurements, a minimum of five body hair samples were collected from each area, which ranged from grades 1 - 4 according to the Ferriman-Gallwey scale. Hair was clipped from the skin surface with scissors. A single measurement of diameter is open to error as the hair varies in its three-dimensional structure. Hence a minimum of 5 hair shafts in each area was evaluated. The diameters were found from their widest portions and then their average diameters were calculated. The measurements were given in μm by the same person under magnification (the diameter of the ocular micrometer is 0.5 mm under 40×10 magnification) with an Olympus BH_2 microscope, and Olympus WHK $10\times/20$ L eyepieces and ocular micrometer (Olympus Corporation, Tokyo, Japan). The sum of the hair diameters on all areas has been termed the "total hair diameter", and the sum of the hair diameters on all sites, excluding the forearm and leg, has been termed the "hormonal hair diameter".

All patients were evaluated by anthropometric measurements: body mass index (BMI) and waist-to-hip ratio (W/H). The body mass index is the ratio of weight (kg) divided by the h^2 (in metric units). Women with BMI < 25 kg/m^2 were classified as normal, while women with BMI $\geq$ 25 kg/m^2, BMI < 30 kg/m^2 were classified as overweight, and women with BMI $\geq$ 30 kg/m^2 were classified as obese.[7] The waist-to-hip (W/H) ratio was used to define these three types of regional fat distribution in the subjects. Women with W/H > 0.86 were classified as android, women with W/H $\leq$ 0.76 were classified as gynecoid, and women with W/H > 0.76 or W/H $\leq$ 0.86 were classified as uniform.[8] The skin color was described according to the Fitzpatrick skin type (Table 1).[9]

Blood samples were taken between 8 - 9 A.M. after a fasting period of 8 - 12 hours. In all patients plasma glucose, androgens and liver function tests were determined. Plasma glucose was determined with the standard laboratory glucose-oxidase method (Beckman Auto Analyzer, Beckman In-

Table 1. Skin Typing According to Fitzpatrick's Skin Type

Skin type	Baseline skin color	Sunburn and tanning history
1	Ivory white	Burns easily, strongly; never tans
2	White	Burns easily, tans minimally with difficulty
3	White	Burns moderatly, tans moderatly and uniformly
4	Beige or lightly tanned	Burns minimally, tans easily and moderatly
5	Moderate brown or tanned	Rarely burns, tans profusely (dark brown)
6	Dark brown or black	Never burns, tans profusely (deep brown or black)

struments, California, USA). Free testosterone and androstenedione were measured with the DSL-10-4900 Enzyme Immunoassay Kit from DSL and DSL-10-3800 Enzyme Immunoassay Kit from DSL (Diagnostic System Laboratories, Inc.; Webster, TX, USA), respectively. Serum total testosterone, dehydroepiandrosterone sulfate (DHEAS) and insulin levels were measured by an electrochemiluminescence immunoassay (Elecsys 1010/ 2010 Kit; Roche Diagnostics GmbH, Mannheim, Germany). Insulin resistance is defined as reduced glucose response to a given amount of insulin.[2] Insulin resistance was estimated using the homeostasis model assessment ($HOMA_{IR}$) from the fasting glucose and insulin concentrations.[10] $HOMA_{IR}$ has been commonly used in clinical studies, and recently, it has been employed in a population-based study.[11] $HOMA_{IR}$ assessment is applied by using the formula below:[10]

$HOMA_{IR}$=Fasting insulin (μU/mL) × Fasting glucose (mmol/liter)/22.5.

The data were expressed as a mean ± SD. The Statistical Package for the Social Sciences computer program (version 11.0 SPSS Corp., Chicago, IL, USA) was used for the statistical analysis. Pearson correlation analysis, partial correlation analysis, multiple linear regression analysis, stepwise regression analysis, X^2 test and Fisher's exact test were used. The receiver operating characteristic (ROC) analysis was performed on the resulting data set of hormonal hair scores and hormonal hair diameter. An ROC analysis is a convenient method for describing the accuracy of the value of a diagnostic test (the hormonal hair diameter in this study) predicts the state of the disease. In this study, hirsutism is defined by a hormonal score value ≥ 8. In the ROC curve, the sensitivity (true positive rate) of disease detection is plotted on the vertical axis and the false positive rate (1-specificity) is plotted on the horizontal axis of the diagram. Significant differences were accepted at $p<0.05$.

RESULTS

The average age of the patients were 35.7 ± 8.5 years. The characteristics of the study population are shown in Table 2. 10.8% of the women had acne. The distribution of the cases according to BMI was: normal weight, 36.8%; overweight, 33.3%; and obese, 29.9%. The distribution of women according to W/H was: android, 17.2%; uniform, 56.8%; and gynecoid, 26.0%. Of all the cases, according to the Fitzpatrick's skin type, 13.7% were in group 2, 42.7% were in group 3,

Table 2. Characteristics of the Study Population

Parameter	Mean ± SD
Age (Year)	35.7 ± 8.5
Gravidity (n)	3.5 ± 2.5
Parity (n)	2.6 ± 1.8
Body Mass Index (kg/m^2)	27.62 ± 5.4
Waist/Hip ratio	0.80 ± 0.07
Testosterone (ng/dL)	41.12 ± 17.16
Free Testosterone (pg/mL)	2.03 ± 0.98
Androstenedione (ng/mL)	1.73 ± 1.03
Dehydroepiandrosterone sulfate (mg/dL)	217.42 ± 92.34
$HOMA_{IR}$	2.54 ± 1.69
Total hair score	5.2 ± 3.6
Hormonal hair score	3.1 ± 2.7
Neutral hair score	2.1 ± 1.4
Total hair diameter (μm)	191.9 ± 90.5
Hormonal hair diameter (μm)	121.8 ± 75.9

and 32.8% were in group 4, 10.8% were in group 5. The percentile values 90%, 95% and 97.5% of the neutral, hormonal, and total hair scores, total and hormonal hair diameter are shown in Table 3. The percentage incidences of the neutral and hormonal scores are represented in Fig. 1 and 2. The 2 figures had a different index distribution.

Table 4 shows the correlations among total, hormonal, and neutral hair scores, total and hormonal hair diameter with age, BMI, W/H, serum total testosterone, free testosterone, androstenedione, DHEAS levels and $HOMA_{IR}$. A hormonal score of 8 or more has been accepted to represent hirsutism,[6] according to this data 8.3% of women had hirsutism. There was a significant relationship between hirsutism and acne, but no significant relationship existed between hirsutism and skin type (in groups 2, 3 and 4-5) (p=0.024 and $p>0.05$, respectively). DHEAS decreased with age (r=-0.277, p=0.032). Age was inversely related to the presence of acne (r=-0.328, $p<0.001$). Testosterone showed a significant positive correlation with DHEAS and free testosterone (r=0.593, $p<0.001$ and r=0.299, p=0.021, respectively). DHEAS showed a significant positive correlation with androstenedione, but did not correlated with $HOMA_{IR}$ (r=0.364, p=0.004 and r=-0.054, $p>0.05$, respectively). To examine the relationship between hair scores and age, partial correlations were performed among total, hormonal, and neutral scores, total, and hormonal hair diameter and age after the correction was made for total, and free testosterone, androstenedione, DHEAS and $HOMA_{IR}$. Analyses were done because androgens and insulin resistance are known to influence age[12,13] There was a significant negative correlation between total, hormonal, and neutral scores, hormonal hair diameter and age after controlling for androgens and $HOMA_{IR}$ (r=-0.432, p=0.001; r=-0.324, p=0.017; r=-0.362, p=0.007; and r=-0.272, p=0.047, respectively), except total hair diameter and age (r=-0.258, p=0.06). In particular total hair scores and neutral scores were affected with age after controlling for androgens and

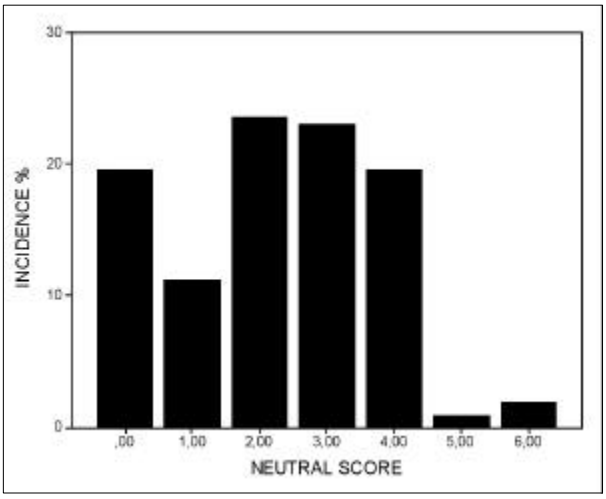

Fig. 1. Percentage of incidence of neutral scores.

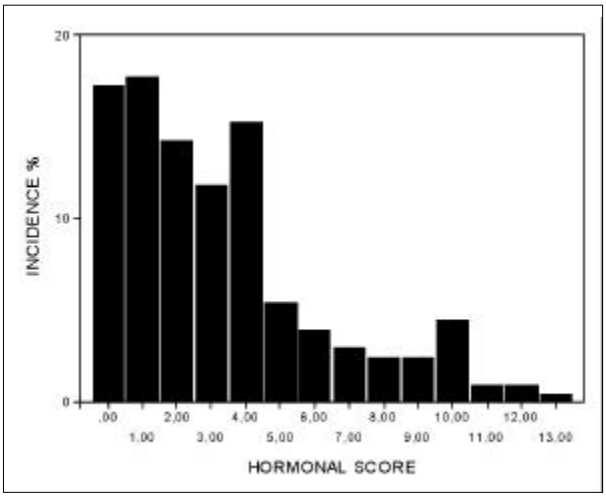

Fig 2. Percentage of incidence of hormonal scores.

Table 3. The Percentile Values 90%, 95% and 97.5% of the Total, Hormonal, Neutral Scores, the Total Hair Diameter and Hormonal Hair Diameter

	90%	95%	97.50%
Total hair score	10.5	11	12
Hormonal hair score	7	9	10
Neutral hair score	4	4	4
Total hair diameter (μm)	300.6	357.2	484.9
Hormonal hair diameter (μm)	216.4	310.2	342.2

Table 4. Correlation Coefficient and *p* Values in Correlation Analysis of Hair Scores, Total and Hormonal Hair Diameter with Clinical Features

		Total hair score	Hormonal hair score	Neutral hair score	Total hair diameter (μm)	Hormonal hair diameter (μm)
Age (year)	r	-0.272	-0.23	-0.257	-0.321	-0.336
	p	<0.001	0.001	<0.001	0.012	0.009
Body Mass Index (kg/m^2)	r	-0.016	0.023	-0.087	-0.082	-0.044
	p	NS	NS	NS	NS	NS
Waist/Hip ratio	r	-0.069	-0.022	-0.136	0.097	0.123
	p	NS	NS	NS	NS	NS
Testosterone (ng/dL)	r	0.291	0.287	0.127	0.27	0.259
	p	0.025	0.027	NS	0.039	0.047
Free Testosterone (pg/mL)	r	0.359	0.294	0.308	0.293	0.239
	p	0.005	0.023	0.016	0.023	NS
Androstenedione (ng/mL)	r	0.422	0.421	0.17	0.393	0.333
	p	0.001	0.001	NS	0.002	0.009
Dehydroepiandrosterone sulfate (μg/dL)	r	0.404	0.347	0.307	0.361	0.35
	p	0.001	0.007	0.017	0.005	0.006
$HOMA_{IR}$	r	0.248	0.293	-0.014	0.344	0.319
	p	NS	0.023	NS	0.007	0.013

NS, No Significant.

Table 5. Partial Regression Coefficient and *p* Value of Total, Hormonal, Neutral Scores, Total Hair Shaft Diameter and Hormonal Hair Diameter with Testosterone, Free Testosterone, Androstenedione, DHEAS and $HOMA_{IR}$ in Multiple Linear Regression Analysis

		Total hair score	Hormonal hair score	Neutral hair score	Total hair diameter (μm)	Hormonal hair diameter (μm)
Testosterone (ng/dL)	β	0.02	0.03	-0.01	0.58	0.5
	p	NS	NS	NS	NS	NS
Free Testosterone (pg/mL)	β	1.132	0.741	0.391	21.591	13.488
	p	0.007	0.044	0.017	NS	NS
Androstenedione (ng/mL)	β	0.974	0.911	0.064	21.922	13.364
	p	0.02	0.015	NS	NS	NS
Dehydroepiandrosterone sulfate (μg/dL)	β	0.008	0.004	0.004	0.179	0.175
	p	NS	NS	NS	NS	NS
$HOMA_{IR}$	β	0.406	0.422	-0.016	16.265	13.344
	p	NS	0.04	NS	0.01	0.017

NS, No Significant.

$HOMA_{IR}$.

Table 5 shows the partial regression coefficient and the *p* value of total, hormonal, and neutral scores, total and hormonal hair diameter with total testosterone, free testosterone, androstenedione, DHEAS and $HOMA_{IR}$ in multiple linear

regression analysis. To further evaluate the interrelationship between these variables, stepwise regression analyses were performed. The total hair score was most strongly predicted by free testosterone and androstenedione (β=1.366, p=0.001 and β=1.391, $p<0.001$, respectively); hormonal hair score by free testosterone and androstenedione (β=0.963, p=0.009 and β=1.204, p=0.001, respectively); neutral score by free testosterone and DHEAS (β=0.346, p=0.027; β=0.0033, p=0.040, respectively); total hair diameter by free testosterone, androstenedione, DHEAS and $HOMA_{IR}$ (β=23.528, p=0.031; β=20.024, p=0.067; β=0.247, p=0.042; and β=16.247, p=0.010, respectively) and hormonal hair diameter by DHEAS and $HOMA_{IR}$ (β=0.307, p=0.002 and β=15.404, p=0.005, respectively).

The correlation between total hair diameter and total hair score was statistically significant (r=0.704 $p<0.001$; regression equation, total hair diameter (μm)=72.827 + 18.513 × total hair score). Also, the correlation between hormonal hair diameter and hormonal hair score was statistically significant (r=0.724, $p<0.001$; regression equation, hormonal hair diameter (μm)=50.255 + 18.497 × hormonal hair score) (Fig. 3).

To determine whether there was a critical value of hormonal hair diameter that could diagnose hirsutism, an ROC curve was generated (Fig. 4). When a hormonal hair diameter ≥ 230.0 μm was chosen as the diagnostic criterion for the prediction of hirsutism (hormonal score ≥ 8), these data yielded a sensitivity of 62.5% and a specificity of 100%). When a hormonal hair diameter ≥91.87 μm was chosen, these data yielded a sensitivity of 100% and a specificity of 42.3% (The area under the curve of the hormonal hair diameter: 0.919, standard error: 0.067). The hormonal hair diameter with the highest percent of true positives and true negatives was 185.5 μm (sensitivity 87.5%, specificity 94.2).

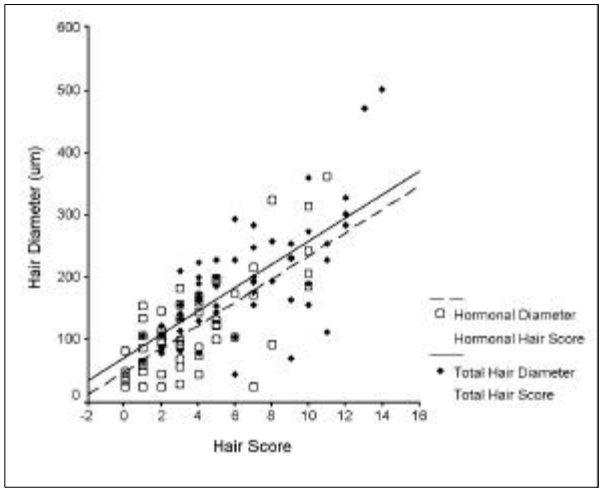

Fig 3. The relationship between total hair diameter and total hair score and the relationship between hormonal hair diameter and hormonal hair score.

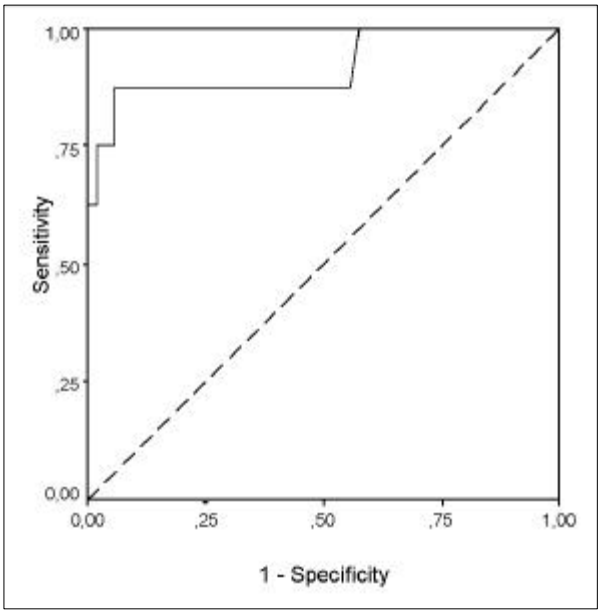

Fig 4. The receiver operator curve analysis of hormonal hair diameter for the diagnosis of hirsutism.

DISCUSSION

95% value for total hair scores was 11, and for the hormonal score, it was 9. As it is known, when evaluating hirsutism, family history and race must be considered. In Turkey and in other Mediterranean nations, there is more hair growth. Atasu T et al. give similar values as in this study (10-14 points) according to the total hair scores in a Turkish population.[14] Ferriman and Gallwey stated that if only the nine "hormonal" skin are considered, 9.9% of their 161 women from the ages of 18 to 38 would have a score above 5, 4.3% would have a score above 7, and 1.2% would have a score greater than 10. So a hormonal score of 8

or more has been accepted as the cut off value for hirsutism.[6] The amount of hair growth is decreased in Orientals compared to Caucasian women and more growth in Mediterranean women than in Nordic women.[2] Hair growth differences between races probably reflect hair follicle differences in 5α-reductase activity.[15] Although racial/ethnic differences in the number, distribution, and androgen sensitivity of hair follicles in normal individuals have been defined, there was no difference in the prevalence of hirsutism in reproductive-aged black and white women.[5,16] In this study, hirsutism did not relate to skin type. Ferriman- Gallwey found, like this study, that hair scores diminished as age increased. They also found a correlation between hormonal and indifferent scores. They also explained that 2 factors underlied body hair growth. One type of growth, with main expression on the forearm and leg may be sexually indifferent and protective in nature. Other hair growth may be related to blood hormone levels or to hair follicle sensitivity to the circulating hormone. The two factors must overlap.[6] Barth et al. thought that forearm hair was androgen-dependent.[17] In the present study, findings were similar to the studies of Ferriman- Gallwey. There was a correlation between hormonal and indifferent scores. These scores had a different incidence of distribution and may be affected by different factors. Blood hormone levels could have less of an effect on the neutral score than on the hormonal score.

The relationship between acne and hirsutism is due to an androgen-induced over secretion by sebaceous glands.[2] In this study, acne correlates with hirsutism. Cibula et al. reported that hirsutism was documented in 21% of the subjects, along with elevated levels of at least one androgen in 81% of the subjects, and polycystic ovaries were found in 50% of women with acne. A positive correlation between the grade of acne severity and any laboratory markers of androgenicity (testosterone, androstenedione, dehydroepiandrosterone, dehydroepiandrosterone sulphate and sex hormone binding globulin) was not demonstrated.[18] This suggests that, in most cases, factors other than hyperandrogenemia are necessary for the development of acne.[19] Acne initiation in childhood has been linked to rising serum levels of dehydroepiandrosterone sulfate. Hirsutism has been correlated with increased levels of serum androgens, notably free testosterone.[20] Slayden et al. found that nonhirsute patients with acne demonstrated significantly lower levels of SHBG and higher free testosterone and DHEAS levels than those in a control group. 63% of acneic patients had at least one androgen value that was above 95% of the controls. In patients of ages 12 - 18 years, 88% had at least one increased androgen value, compared with 55% of the patients of ages 19 - 43 years. Hyperandrogenemia was evident in a majority of nonhirsute acneic patients studied, regardless of age.[21] Acne is a sign of increased androgen activity. Up to 60% of women with acne have normal circulation levels of androgen. They display increased 5 α-reductase activity in the piloseboceous unit, and these women benefit from the androgen suppressive treatment.[2,22]

In the present study, obesity (according to the BMI) did not affect hair scores. Several studies have shown that an increase in W/H ratio may be a manifestation of endocrine and metabolic changes. Plasma androgens are an important determinant of body fat topography in premenopausal women. In turn, an android distribution of adipose tissue is frequently associated with metabolic disorders. In the hirsute subjects, upper body fat predominance was determined.[23,24] Meirow et al. found that in polycystic ovary syndrome, the insulin resistant patients were significantly more obese (higher BMI and W/H ratio) and more hirsute than the insulin non-resistant patients'.[25] In this study, there was no relationship the W/H ratio with hair scores and total hair diameter in healthy women.

The primary factor in hirsutism is elevated androgen levels (usually testosterone) that produces an initial growth stimulus and then sustains continued growth. Essentially, women with hirsutism will have an increased production rate of testosterone and androstenedione.[26] DHEAS serves as a prehormone in hair follicles, providing substrate for the hair follicle synthesis of androgens. Hence, elevated DHEAS levels contribute to the clinical problem of hirsutism.[27]

Circulating levels of DHEAS decreased with age. It further suggested that the age-related decline may be related to an increased resistance to

insulin.[13] In anovulatory women a simple inverse relationship between insulin and adrenal androgen levels does not exist.[28] In this study, DHEAS decreased with age. There was no relationship between $HOMA_{IR}$ and DHEAS. Hyperandrogenism and hyperinsulinemia are commonly associated.[2] In many women, a disorder in insulin action precedes the increase in androgens. Hyperinsulinemia can directly augment thecal cell androgen production in the ovary, and also, hyperinsulinemia contributes to the hyperandrogenism by inhibiting hepatic synthesis of sex hormone-binding globulin and insulin-like growth factor binding protein-1, actions that increase free testosterone levels and augment IGF-I stimulation of thecal androgen synthesis, respectively.[2] Sex steroids, a number of local and systemic factors (such as various growth factors, cytokines, thyroid and growth hormone) and 5α-reductase activity can act directly and indirectly on the hair growth.[5] Dihydrotestosterone is the major nuclear androgen in many sensitive tissues. 3 α-Androstanediol is the peripheral tissue metabolite of dihydrotestosterone. 3 α-androstanediol glucuronide correlates with the level of 5 α-reductase activity in the skin. Measurement of 3 α-androstanediol glucuronide is not used routinely in clinical approaches because of two reasons. First, values of hirsute women overlap the normal range by about 20%. Second, the ultimate diagnosis and treatment are not affected by this test.[2]

A relationship between hair scores, which are semi-quantitative, and hair shaft diameter, which is more objective, was found. Even hair in the same area have different diameter, so the need for multiple hair samples and multiple measurements make this method impractical. A study evaluating hair shaft diameter and hair score was not found in the literature. Hair shaft diameter measurement can be performed specific cases. The Ferriman-Gallwey scoring system is reserved for studies of hirsutism, but even for this purpose, it is limited by subjective variability.[2]

By reviewing the ROC curve, one could make an argument for a critical cut-off anywhere between 91.87 μm (sensitivity 100%) and 230.0 μm (specificity 100%). Additional studies might help to solve this specific problem.

In conclusion, the hair scores and diameters showed meaningful positive correlation with androgen levels and negative correlation with age. There was a significant negative correlation between total, hormonal, neutral scores, hormonal hair diameter and age after controlling for androgens and $HOMA_{IR}$ except total hair diameter. In particular, total hair scores and neutral scores were affected with age after controlling for androgens and $HOMA_{IR}$.

There was a positive correlation between total hair shaft diameter and total hair score and also between hormonal hair diameter and hormonal hair score.

Neutral score was correlated especially with free testosterone and DHEAS, but less hormone dependent than hormonal score. While total and hormonal hair scores might be strongly affected by androgens, hair diameters might be affected by $HOMA_{IR}$ and androgens.

In this study, 95% value for total hair scores was 11, and this value of hormonal scores was 9.

REFERENCES

1. Hershlag A, Peterson CM. Endrocrine disorders. In: Berek JS, Adashi EY, Hillard PA, editors. Novak's gynecology. 12th ed. Baltimore, Maryland: Williams & Wilkins Baltimore; 1996. p.833-86.
2. Hirsutism. In: Sperof L, Glass RH, Kase NG, editors. Clinical gynecologic endocrinology and infertility. 6th ed. Baltimore, Marland: Lippincott Williams & Wilkins Baltimore; 1999. p.523-56.
3. Hatch R, Rosenfield RL, Kim MH, Tredway D. Hirsutism: Implications, etiology, and management. Am J Obstet Gynecol 1981;140:815-30.
4. Young R, Sinclair R. Hirsutes. I: Diagnosis. Australas J Dermatol 1998;39:24-8.
5. Azziz R, Carmina E, Sawaya ME. Idiopathic hirsutism. Endocr Rev 2000;21:347-62.
6. Ferriman D, Gallwey JD. Clinical assessment of body hair growth in women. J Clin Endocrinol Metab 1961; 21:1440-7.
7. Obesity. In: Speroff L, Glass RH, Kase NG, editors. Clinical gynecologic endocrinology and infertility. 6th ed. Baltimore, Marland: Lippincott Williams & Wilkins Baltimore; 1999. p.781-808.
8. Presta E, Leibel RL, Hirsch J. Regional changes in adrenergic receptor status during hypocaloric intake do not predict changes in adipocyte size or body shape. Metabolism 1990;39:307-15.
9. Pathak MA, Nghiem P, Fitzpatrick TB. Acute and chronic effects on the sun. In: Freedberg IM, Eisen AZ,

Wolf K, Austen KF, Goldsmith LA, Katz SI, et al, editors. Fitzpatric's Dermatology in general medicine. 5th ed. New York: McGraw-Hill; 1999. p.1598-607.
10. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RL. Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985;28:412-9.
11. Haffner SM, Miettinen H, Stern MP. The homeostasis model in the San Antonio Heart Study. Diabetes Care 1997;20:1087-92.
12. Labrie F, Belanger A, Cusan L, Gomez JL, Candas B. Marked decline in serum concentrations of adrenal C19 sex steroid precursors and conjugated androgen metabolites during aging. J Clin Endocrinol Metab 1997;82: 2396-402.
13. Liu CH, Laughlin GA, Fischer UG, Yen SS. Marked attenuation of ultradian and circadian rhythms of dehydroepiandrosterone in postmenopausal women: evidence for a reduced 17,20-desmolase enzymatic activity. J Clin Endocrinol Metab 1990;71:900-6.
14. Atasü T. Kadında androjen metabolizması bozuklukları ve görülen klinik tablolar (Hirsutism, Virilism). In: Atasü T, Şahmay S, editors. Reprodüktif Endokrinoloji. Istanbul: Logos; 1990. p.277-313.
15. Lookingbill DP, Demers LM, Wang C, Leung A, Rittmaster RS, Santen RJ. Clinical and biochemical parameters of androgen action in normal and healthy Caucasian versus Chinese subjects. J Clin Endocrinol Metab 1991;72:1242-8.
16. Knochenhauer ES, Key TJ, Kahsar-Miller M, Waggoner W, Boots LR, Azziz R. Prevalence of the polycystic ovarian syndrome in unselected black and white women of the Southeastern United States: a prospective study. J Clin Endocrinol Metab 1998;83:3078-82.
17. Barth JH, Cherry CA, Wojnarowska F, Dawber RPR. Spironolactone is an effective and well tolerated systemic antiandrogen therapy for hirsute women. J Clin Endocrinol Metab 1989;68:966-70.
18. Cibula D, Hill M, Vohradnikova O, Kuzel D, Fanta M, Zivny J. The role of androgens in determining acne severity in adult women. Br J Dermatol 2000;143:399-404.
19. Sheehan-Dare RA, Hughes BR, Cunliffe WJ. Clinical markers of androgenicity in acne vulgaris. Br J Dermatol 1988;119:723-30.
20. Lucky AW. Hormonal correlates of acne and hirsutism. Am J Med 1995;98(1A):89S-94S.
21. Slayden SM, Moran C, Sams WM Jr, Boots LR, Azziz R. Hyperandrogenemia in patients presenting with acne. Fertil Steril 2001;75:889-92.
22. Carmina E, Lobo RA. Evidence for increased androsterone metabolism in some normoandrogenic women with acne. J Clin Endocrinol Metab 1993;76:1111-4.
23. Evans DJ, Barth JH, Burke CW. Body fat topography in women with androgen excess. Int J Obes 1988;12: 157-62.
24. Hauner H, Ditschuneit HH, Pal SB, Pfeiffer EF. Distribution of adipose tissue and complications of obesity in overweight women with and without hirsutism. Dtsch Med Wochenschr 1987;112:709-13.
25. Meirow D, Yossepowitch O, Rösler A, Brzezinski A, Schenker JG, Laufer N, et al. Insulin resistant and non-resistant polycystic ovary syndrome represent two clinical and endocrinological subgroups. Hum Reprod 1995;10:1951-6.
26. Bardin CW, Lipsett MB. Testosterone and androstenedione blood production rates in normal women and women with idiopathic hirsutism or polycystic ovaries. J Clin Invest 1967;46:891-902.
27. Haning RV Jr, Hackett RJ, Flood CA, Loughlin JS, Zhao QY, Longcope C. Plasma dehydroepiandrosterone sulfate serves as a prehormone for 48% of follicular fluid testosterone during treatment with menotropins. J Clin Endocrinol Metab 1993;76:1301-7.
28. Alper MM, Garner PR. Elevated serum dehydroepiandrosterone sulfate levels in patients with insulin resistance, hirsutism, and acanthosis nigricans. Fertil Steril 1987;47:255-8.